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=> d his

     (FILE 'HOME' ENTERED AT 08:02:19 ON 28 FEB 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:02:45 ON 28 FEB 2006
L1        517312 S CELL (W) PROLIFERAT?
L2        281202 S ATHEROSCLEROSIS
L3          9346 S L1 AND L2
L4         72799 S SIALIC (W) ACID
L5            31 S L3 AND L4
L6            13 DUP REM L5 (18 DUPLICATES REMOVED)
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                   IPC reform
 NEWS  8  DEC 23   New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                   USPAT2
 NEWS  9  JAN 13   IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS 10  JAN 13   New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                   INPADOC
 NEWS 11  JAN 17   Pre-1988 INPI data added to MARPAT
 NEWS 12  JAN 17   IPC 8 in the WPI family of databases including WPIFV
 NEWS 13  JAN 30   Saved answer limit increased
 NEWS 14  JAN 31   Monthly current-awareness alert (SDI) frequency
                   added to TULSA
 NEWS 15  FEB 21   STN AnaVist, Version 1.1, lets you share your STN AnaVist
                   visualization results
 NEWS 16  FEB 22   Status of current WO (PCT) information on STN
 NEWS 17  FEB 22   The IPC thesaurus added to additional patent databases on STN
 NEWS 18  FEB 22   Updates in EPFULL; IPC 8 enhancements added

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               AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
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=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
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                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.21       0.21

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FILE 'LIFESCI' ENTERED AT 08:02:45 ON 28 FEB 2006
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=> s cell (w_proliferat?
MISSING OPERATOR 'CELL (W_PROLIFER'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s cell (w) proliferat?
L1      517312 CELL (W) PROLIFERAT?

=> s atherosclerosis
L2      281202 ATHEROSCLEROSIS

=> s l1 and l2
L3        9346 L1 AND L2

=> s sialic (w) acid
L4       72799 SIALIC (W) ACID

=> s l3 and l4
L5          31 L3 AND L4

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6           13 DUP REM L5 (18 DUPLICATES REMOVED)

=> d 1-13 ibib ab

L6   ANSWER 1 OF 13  SCISEARCH  COPYRIGHT (c) 2006 The Thomson Corporation  on
     STN
ACCESSION NUMBER:    2006:134062  SCISEARCH
THE GENUINE ARTICLE: 006GD
```

TITLE: Colominic acid inhibits the proliferation of cultured bovine aortic endothelial cells and injures their monolayers: Cell density-dependent effects prevented by sulfation

AUTHOR: Yamamoto C; Morita Y; Yamaguchi S; Hayashi T; Kaji T (Reprint)

CORPORATE SOURCE: Hokuriku Univ, Fac Pharmaceut Sci, Dept Environm Hlth, Ho 3 Kanagawa machi, Kanazawa, Ishikawa 9201181, Japan (Reprint); Hokuriku Univ, Fac Pharmaceut Sci, Dept Environm Hlth, Kanazawa, Ishikawa 9201181, Japan; Marukin Bio Inc, Uji, Kyoto 6110013, Japan; Toyama Med & Pharmaceut Univ, Fac Pharmaceut Sci, Dept Pharmacognosy, Toyama 9300194, Japan
t-kaji@hokuriku-u.ac.jp

COUNTRY OF AUTHOR: Japan

SOURCE: LIFE SCIENCES, (18 JAN 2006) Vol. 78, No. 8, pp. 844-850. ISSN: 0024-3205.

PUBLISHER: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 41

ENTRY DATE: Entered STN: 9 Feb 2006
Last Updated on STN: 9 Feb 2006

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Colominic acid (CA), produced by Escherichia coli K1, is a polymer of **sialic acid** linked through alpha (2 -> 8) glycosidic linkages. Although there are several studies on the biological activities of chemically sulfated CA, the activity of CA has been incompletely understood. In the present study, we investigated the effects of CA, prepared as an alpha 2,8-linked homopolymer of N-acetylneuraminic acid, on the proliferation and monolayer maintenance of bovine aortic endothelial cells in culture. The results indicate that CA potently inhibits the proliferation of sparse endothelial cells without nonspecific cell damage. The inhibitory effect of CA was markedly stronger than those of sodium spirulan and calcium spirulan, known polysaccharides that inhibit endothelial **cell proliferation**. On the other hand, in dense endothelial cells, CA induced nonspecific cell damage and markedly injured the monolayer. These results indicate that CA has two distinct effects on vascular endothelial cells: one is the inhibition of proliferation when the cell density is low, and the other is the nonspecific cytotoxicity when the cell density is high. Interestingly, these cell density-dependent effects of CA could be prevented by sulfation of the CA chains. Therefore, it is concluded that CA not only inhibits the proliferation of sparse endothelial cells without nonspecific cell damage but also injures dense cells in a monolayer by nonspecific cytotoxicity, which can be prevented by sulfation of the polysaccharide.

L6 ANSWER 2 OF 13 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2005285001 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15930733

TITLE: Inhibition of cultured bovine aortic smooth muscle **cell proliferation** by colominic acid.

AUTHOR: Yamamoto Chika; Yamaguchi Shinya; Hayashi Toshimitsu; Kaji Toshiyuki

CORPORATE SOURCE: Department of Environmental Health, Faculty of Pharmaceutical Sciences, Hokuriku University, Kanazawa, Japan.

SOURCE: Biological & pharmaceutical bulletin, (2005 Jun) Vol. 28, No. 6, pp. 994-7.
Journal code: 9311984. ISSN: 0918-6158.

PUB. COUNTRY: Japan

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200601
ENTRY DATE: Entered STN: 20050603
Last Updated on STN: 20060120
Entered Medline: 20060119

AB Colominic acid (CA) is an alpha2,8-linked polymer of **sialic acid**, originally isolated from capsular Escherichia coli K1. Since inhibition of arterial smooth muscle cell hyperplasia is one of the effective strategies to prevent **atherosclerosis**, we investigated the effect of CA, purified as an alpha2,8-linked homopolymer of N-acetylneuraminic acid, on the proliferation of bovine aortic smooth muscle cells in culture. The results demonstrate that CA inhibits the proliferation of the cells without nonspecific cell damage. Sulfation did not modify the inhibitory effect of CA. Specifically, the inhibitory effect of sulfated CA was almost equal to that of CA in vascular smooth muscle **cell proliferation**. On the other hand, it was suggested that the inhibition of the proliferation by CA is in a degree similar to that by heparin but weaker than that by sodium/calcium-spirulans, known sulfated polysaccharides as the potent inhibitor of vascular smooth muscle cells. The present data suggest that CA with or without sulfate groups can be an origin of beneficial agents that prevents **atherosclerosis** through a moderate inhibition of arterial smooth muscle **cell proliferation**.

L6 ANSWER 3 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 2

ACCESSION NUMBER: 2005328787 EMBASE
TITLE: **Sialic acid** as a protective barrier against neointima development.
AUTHOR: Cuniberti L.A.; Martinez V.; Schachter J.; Magarinos G.; Meckert P.C.; Laguens R.P.; Levenson J.; Werba J.P.
CORPORATE SOURCE: L.A. Cuniberti, Lipid and Atherosclerosis Research Laboratory, Department of Pathology, Favaloro University, Buenos Aires, Argentina. cuniberti@favaloro.edu.ar
SOURCE: Atherosclerosis, (2005) Vol. 181, No. 2, pp. 225-231. .
Refs: 30
ISSN: 0021-9150 CODEN: ATHSBL
PUBLISHER IDENT.: S 0021-9150(05)00088-2
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050818
Last Updated on STN: 20050818

AB Arterial **sialic acid** (SA) has been shown to attenuate the binding of fibrinogen and low-density lipoproteins (LDL) to the vessel wall, presumably protecting against **atherosclerosis**. This study was aimed to assess the effect of changes in SA content in intimal thickening, an early step in the development of **atherosclerosis**. New Zealand white rabbits were subjected to bilateral carotid periarterial collaring, followed by in situ-perfusion with neuroaminidase (random artery) and with vehicle (contralateral control artery). The efficiency of SA removal was evaluated in perfusates and arterial homogenates, and arterial tissue samples were obtained 7 and 14 days after the intervention to assess morphological changes. Neuraminidase significantly reduced SA by 16.7%. Arterial desialylation was associated with a significantly increased neointimal formation. Proliferation of smooth muscle cells (SMCs), assessed by incorporation of bromo-2'-deoxyuridine into replicating DNA was also significantly increased in desialylated arteries. In addition, immunohistochemical studies showed a slightly stronger oxidized-LDL (ox-LDL) immunostaining in neointima of desialylated arteries

than in control vessels. A mild reduction of SA increases intimal thickening, at least partly due to an enhanced proliferation of SMCs, and may facilitate the accretion of atherogenic lipoproteins, providing evidence for the potential role of SA in the protection against neointimal development. .COPYRGT. 2005 Elsevier Ireland Ltd. All rights reserved.

L6 ANSWER 4 OF 13 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2004382649 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15175338
TITLE: Disialoganglioside (GD3) synthase gene expression suppresses vascular smooth muscle cell responses via the inhibition of ERK1/2 phosphorylation, cell cycle progression, and matrix metalloproteinase-9 expression.
AUTHOR: Moon Sung-Kwon; Kim Hong-Man; Lee Young-Choon; Kim Cheorl-Ho
CORPORATE SOURCE: National Research Laboratory for Glycobiology, Ministry of Science and Technology, Dongguk University College of Oriental Medicine, Kyungju City, Kyungbuk 780-714, Korea.
SOURCE: The Journal of biological chemistry, (2004 Aug 6) Vol. 279, No. 32, pp. 33063-70. Electronic Publication: 2004-06-02. Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200410
ENTRY DATE: Entered STN: 20040803
Last Updated on STN: 20041026
Entered Medline: 20041025

AB **Sialic acid**-containing glycosphingolipids (gangliosides) have been implicated in the regulation of various biological phenomena such as **atherosclerosis**. Recent report suggests that exogenously supplied disialoganglioside (GD3) serves a dual role in vascular smooth muscle cells (VSMC) proliferation and apoptosis. However, the role of the GD3 synthase gene in VSMC responses has not yet been elucidated. To determine whether a ganglioside is able to modulate VSMC growth, the effect of overexpression of the GD3 synthase gene on DNA synthesis was examined. The results show that the overexpression of this gene has a potent inhibitory effect on DNA synthesis and ERK phosphorylation in cultured VSMC in the presence of PDGF. The suppression of the GD3 synthase gene was correlated with the down-regulation of cyclinE/CDK2, the up-regulation of the CDK inhibitor p21 and blocking of the p27 inhibition, whereas up-regulation of p53 as the result of GD3 synthase gene expression was not observed. Consistently, blockade of GD3 function with anti-GD3 antibody reversed VSMC proliferation and cell cycle proteins. The expression of the GD3 synthase gene also led to the inhibition of TNF-alpha-induced matrix metalloproteinase-9 (MMP-9) expression in VSMC as determined by zymography and immunoblot. Furthermore, GD3 synthase gene expression strongly decreased MMP-9 promoter activity in response to TNF-alpha. This inhibition was characterized by the down-regulation of MMP-9, which was transcriptionally regulated at NF-kappaB and activation protein-1 (AP-1) sites in the MMP-9 promoter. Finally, the overexpression of MMP-9 in GD3 synthase transfectant cells rescued VSMC proliferation. However, MMP-2 overexpression was not affected by **cell proliferation**. These findings suggest that the GD3 synthase gene represents a physiological modulator of VSMC responses that may contribute to plaque instability in **atherosclerosis**.

L6 ANSWER 5 OF 13 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
ACCESSION NUMBER: 2004-00309 BIOTECHDS
TITLE: Use of an agent that prevents or inhibits Mycoplasma infection, for manufacturing a medicament for treating or preventing a disorder associated with increased **cell**

proliferation, e.g. atherosclerotic vascular disease or malignancy;
recombinant Trypanosoma cruzi protein application in infection, tumor and vascular disease therapy

AUTHOR: HIGUCHI M D L; SCHENKMAN S
PATENT ASSIGNEE: HIGUCHI M D L; SCHENKMAN S
PATENT INFO: US 2003124109 3 Jul 2003
APPLICATION INFO: US 2002-86913 1 Mar 2002
PRIORITY INFO: BR 2001-2648 3 Jul 2001; BR 2000-2989 3 Jul 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-810968 [76]

AB DERWENT ABSTRACT:

NOVELTY - Use of an agent that prevents or inhibits Mycoplasma infection for manufacturing a medicament for treating a disorder associated with increased **cell proliferation**.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition for treating or preventing Mycoplasma infection in a subject suffering from a disorder associated with increased **cell proliferation** or a co-infection with mycoplasma and a second microbe, comprising an agent that prevents or inhibits **sialic acid**-mediated attachment of mycoplasma to cells of the subject.

BIOTECHNOLOGY - Preferred Composition: The agent is an antibiotic or an enzyme having an activity consisting of neuraminidase and/or trans-sialidase activity. The enzyme is derived from a Trypanosoma cruzi microorganism, where the enzyme is a native or a recombinant enzyme. The enzyme has a fully defined sequence of 669 amino acids given in the specification. A vector containing the DNA insert having a fully defined sequence of 2010 bp given in the specification produces the enzyme.

ACTIVITY - Antibacterial; Antiarteriosclerotic; Cytostatic; Anti-HIV. A 64-year-old female patient with a palpable abdominal mass and a tumoral mass in the rectum was administered 50 ml of native trans-sialidase (TSN) intraperitoneally on alternate days for a period of 14 days. On day 23, with mycoplasmas confirmed in the bone marrow, erythromycin (500 mg/day) was given for a further 20 days. Clinical improvement and normalization of blood leukocytes was seen after 2 days. Considering the important clinical improvement and reduction in abdominal mass, a second session of TSN was administered. The patient demonstrated improvement in general clinical status. Tomography detected a reduction in tumoral mass. Results showed that trans-sialidase is effective as a drug in the treatment of neoplasia, removing mycoplasmas from the neoplastic cells leading to their apoptosis.

MECHANISM OF ACTION - Neuraminidase; Trans-sialidase.

USE - The composition or the agent that prevents or inhibits mycoplasma infection is useful for manufacturing a medicament for treating or preventing a disorder associated with increased **cell proliferation**, e.g. atherosclerotic vascular disease or malignant disease, or a disease associated with co-infection with mycoplasma and a second microbe such as human immunodeficiency virus or a Chlamydia microbe (all claimed).

ADMINISTRATION - The amount of the enzyme administered is about 106-1013 units per day. Administration may be intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous, or intramuscular. (32 pages)

L6 ANSWER 6 OF 13 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2002273319 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11861654
TITLE: GD3 recruits reactive oxygen species to induce **cell proliferation** and apoptosis in human aortic smooth muscle cells.
AUTHOR: Bhunia Anil Kumar; Schwarzmann Gunter; Chatterjee Subroto
CORPORATE SOURCE: Department of Pediatrics, Lipid Research Atherosclerosis Unit, The Johns Hopkins University School of Medicine,

Baltimore, Maryland 21044, USA.
SOURCE: The Journal of biological chemistry, (2002 May 10) Vol. 277, No. 19, pp. 16396-402. Electronic Publication: 2002-02-22.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020517
Last Updated on STN: 20030105
Entered Medline: 20020613

AB **Sialic acid** containing glycosphingolipids (gangliosides) are expressed on the surface of all mammalian cells and have been implicated in regulating various biological phenomena; however, the detailed signaling mechanisms involved in this process are not known. We report here a novel aspect of disialoganglioside, GD3-mediated regulation of **cell proliferation** and cell death via the recruitment of reactive oxygen species (ROS). A low concentration (2.5-10 microm) of GD3, incubated with human aortic smooth muscle cells for a short period of time (10-30 min), stimulates superoxide generation via the activation of both NADPH oxidase and NADH oxidase activity. This leads to downstream signaling leading to **cell proliferation** and apoptosis. However, [(3)H]GD3 incubated with the cells under such conditions was found in a trypsin-sensitive fraction that was separable from endogenous GD3. The exact mechanism causing ROS generation and downstream signaling remains to be elucidated. The uptake of GD3 was accompanied by a 2.5-fold stimulation in the activity of mitogen-activated protein (MAP) kinase and 5-fold stimulation in **cell proliferation**. Preincubation of cells with membrane-permeable antioxidants, pyrrolidine dithiocarbamate, and N-acetylcysteine abrogated the superoxide generation and **cell proliferation**. In contrast, at higher concentrations (50-200 microm) GD3 inhibited the generation of superoxides but markedly stimulated the generation of nitric oxide (NO) (10-fold compared with control). This in turn stimulated mitochondrial cytochrome c release and intrachromosomal DNA fragmentation, which lead to apoptosis. In sum, at a low concentration, GD3 recruits superoxides to activate p44 MAPK and stimulates **cell proliferation**. In contrast, at high concentrations GD3 recruits nitric oxide to scavenge superoxide radicals that triggered signaling events that led to apoptosis. These observations might have relevance in regard to the potential role of GD3 in aortic smooth muscle **cell proliferation** and apoptosis that may contribute to plaque rupture in **atherosclerosis**.

L6 ANSWER 7 OF 13 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:79743 BIOSIS
DOCUMENT NUMBER: PREV200300079743
TITLE: Angiotensin II accelerated **atherosclerosis** is attenuated in osteopontin-deficient mice.
AUTHOR(S): Bruemmer, Dennis [Reprint Author]; Collins, Allan R. [Reprint Author]; Noh, Grace [Reprint Author]; Kintscher, Ulrich; Ozawa, Yuri [Reprint Author]; Fleck, Eckart; Law, Ronald E. [Reprint Author]; Hsueh, Willa [Reprint Author]
CORPORATE SOURCE: Univ of CA, Los Angeles, Los Angeles, CA, USA
SOURCE: Circulation, (November 5 2002) Vol. 106, No. 19 Supplement, pp. II-217-II-218. print.
Meeting Info.: Abstracts from Scientific Sessions. Chicago, IL, USA. November 17-20, 2002. American Heart Association. ISSN: 0009-7322 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English

ENTRY DATE: Entered STN: 6 Feb 2003
Last Updated on STN: 6 Feb 2003

L6 ANSWER 8 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 5

ACCESSION NUMBER: 2002114908 EMBASE
TITLE: Gangliosides GM1 and GM2 induce vascular smooth muscle **cell proliferation** via extracellular signal-regulated kinase 1/2 pathway.
AUTHOR: Gouni-Berthold I.; Seul C.; Ko Y.; Hescheler J.; Sachinidis A.
CORPORATE SOURCE: Dr. A. Sachinidis, Institute of Neurophysiology, University of Cologne, Robert-Koch Str. 39, 50931 Cologne, Germany. A.Sachinidis@uni-koeln.de
SOURCE: Hypertension, (2001) Vol. 38, No. 5, pp. 1030-1037. .
Refs: 52
ISSN: 0194-911X CODEN: HPRTDN
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020418
Last Updated on STN: 20020418

AB Gangliosides, **sialic acid**-containing glycophospholipids, accumulate in atherosclerotic vessels and appear to regulate the proliferation of various cell types. Furthermore, vascular smooth muscle cell (VSMC) proliferation is associated with the development and progression of cardiovascular diseases. To demonstrate whether gangliosides are able to modulate the VSMC growth, the effect of gangliosides GM1, GM2, and GM3 on cell DNA synthesis and cell number has been examined. Moreover, we investigated possible intracellular mechanisms by which GM1 and GM2 elicit their mitogenic effects. Stimulation of VSMCs with GM1 and GM2 resulted in a dose-dependent increase in DNA synthesis and cell number, whereas GM3 caused a decrease in DNA synthesis. GM1 and GM2 (50 μmol/L) stimulate phosphorylation of extracellular signal-regulated kinases (ERKs) 1 and 2 and phosphorylation of the c-Jun N-terminal kinase (JNK), with a maximum at 15 minutes, but they do not have an effect on the phosphorylation of p38 mitogen-activated protein kinase (MAPK). GM3 (50 μmol/L), on the other hand, does not stimulate any of the 3 aforementioned MAPKs. Pretreatment of the cells with 20 μmol/L PD 098,059 caused a complete inhibition of ERK1/2 and JNK MAPK, whereas pretreatment with a Ras (farnesyl transferase) inhibitor did not abrogate the GM1- and GM2-induced ERK1/2 phosphorylation. Furthermore, GM1 and GM2 did not activate Raf-1 kinase. Interestingly, pretreatment of VSMCs with 100 nmol/L pertussis toxin resulted in a complete inhibition of the ERK1/2 phosphorylation. Finally, the GM1- and GM2-induced increase in cell number was significantly inhibited by PD 098,059. We may conclude that GM1 and GM2 stimulate ERK1/2 via a pertussis toxin-sensitive G(i)-coupled receptor through a Raf-I kinase-independent pathway. Moreover, the GM1- and GM2-induced VSMC growth is ERK1/2 dependent.

L6 ANSWER 9 OF 13 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:723793 HCAPLUS
DOCUMENT NUMBER: 130:484
TITLE: Use of sialyl galactosides and related compounds as antiangiogenic agents
INVENTOR(S): Defrees, Shawn; Bayer, Robert J.; Ratcliffe, Murray
PATENT ASSIGNEE(S): Cytel Corp., USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9848817	A1	19981105	WO 1998-US8932		19980501
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
AU 9872777	A1	19981124	AU 1998-72777		19980501
PRIORITY APPLN. INFO.:			US 1997-45271P	P	19970501
			US 1998-71140	A2	19980430
			WO 1998-US8932	W	19980501

OTHER SOURCE(S): MARPAT 130:484

AB The present invention provides methods and compns. for inhibiting proliferation of endothelial cells by contacting the cells with a **sialic acid** or a sialyl glycoside. The methods are useful for treating conditions that are characterized by undesirable cellular proliferation such as neoplasia, metastasis, restenosis, retinopathy, **atherosclerosis**, vascular diseases, skin diseases.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 13 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1995:698893 HCAPLUS
DOCUMENT NUMBER: 123:74872
TITLE: Action of cell-surface receptors changing the main characteristics of cellular function, and medical applications thereof in **atherosclerosis**, diabetes, cancer, and other disorders
INVENTOR(S): Zagyansky, Yuly
PATENT ASSIGNEE(S): Fr.
SOURCE: Fr. Demande, 309 pp.
CODEN: FRXXBL
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
FR 2711318	A1	19950428	FR 1993-11198	19930921
PRIORITY APPLN. INFO.:			FR 1993-11198	19930921

AB From a general process of activity of cell-surface receptors, the principal dogmas of life sciences are revised and clearly reestablished. As a result, more universal new processes and structures are established, e.g. protein kinase C vesicles, the Ca-K(Ca) wave, propagation of cell signals to DNA, etc. Consequently, the process of creation of basal lamina and organs; the structure of contact inhibition; cardiac, skeletal, and smooth muscle action; cell motility; attachment and penetration of bacterial and viral toxins; etc. have also been clearly established. Mol. origins of, and prepns. against, major disorders (diabetes, dystrophy, scurvy, rickets, etc.) are included. The finished accordance from all the given principles and very different domains again confirms the incontestable validity of findings advanced over a half-century. Included are 44 schematic diagrams and 1514 refs.

L6 ANSWER 11 OF 13 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:196789 HCAPLUS
DOCUMENT NUMBER: 124:220476
TITLE: Depth of the essential characteristics of the signal transmission process starting from the cell surface, and their medicinal applications in **atherosclerosis**, diabetes, cancer, scurvy, rickets, and other conditions
INVENTOR(S): Zagyansky, Yuly
PATENT ASSIGNEE(S): Fr.
SOURCE: Can. Pat. Appl., 320 pp.
CODEN: CPXXEB
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CA 2139676	AA	19951225	CA 1994-2139676	19941025
PRIORITY APPLN. INFO.:			US 1994-281731 A	19940624

AB From a general process of activity of cell-surface receptors, the principal dogmas of life sciences are revised and clearly reestablished. As a result, more universal new processes and structures are established, e.g. protein kinase C vesicles, the Ca-K(Ca) wave, propagation of cell signals to DNA, etc. Consequently, the process of creation of basal lamina and organs; the structure of contact inhibition; cardiac, skeletal, and smooth muscle action; cell motility; attachment and penetration of bacterial and viral toxins; etc. have also been clearly established. Mol. origins of, and prepns. against, a variety of disorders (diabetes, dystrophy, scurvy, rickets, etc.) are included. Creation of twins is described. The finished accordance from all the given principles and very different domains again confirms the incontestable validity of findings advanced over a half-century. Included are 44 schematic diagrams and 1514 refs.

L6 ANSWER 12 OF 13 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 95324077 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7600681
TITLE: Modified (desialylated) low-density lipoprotein measured in serum by lectin-sorbent assay.
AUTHOR: Tertov V V; Sobenin I A; Orekhov A N
CORPORATE SOURCE: Institute of Experimental Cardiology, Cardiology Research Center, Moscow, Russia.
SOURCE: Clinical chemistry, (1995 Jul) Vol. 41, No. 7, pp. 1018-21.
Journal code: 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950822
Last Updated on STN: 19950822
Entered Medline: 19950810

AB Modified low-density lipoprotein (LDL) with a low **sialic acid** content was found in the blood of patients with coronary **atherosclerosis**. This desialylated lipoprotein causes lipid accumulation in arterial smooth-muscle cells and stimulates **cell proliferation** and production of the extracellular matrix, i.e., induces all atherogenic manifestations at the cellular level. We have developed a lectin-sorbent assay for the determination of desialylated LDL in sera. The assay is based on the binding of desialylated LDL by immobilized Ricinus communis agglutinin with subsequent measurement of lipoprotein through use of anti-apolipoprotein (apo) B antibody. The assay is sensitive to desialylated apo B concentrations as low as 5

micrograms/L. The intraassay and interassay CVs were 4.8% and 11.3%, respectively. Comparison between the lectin-sorbent assay and a lectin chromatographic technique showed a good correlation. This determination of modified desialylated LDL in human serum with high accuracy and reproducibility may help establish the diagnostic value of this lipoprotein as a risk factor of **atherosclerosis**.

L6 ANSWER 13 OF 13 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1988:394039 BIOSIS
DOCUMENT NUMBER: PREV198886066678; BA86:66678
TITLE: THROMBOCYTE ADHESION AND ACTIVATION ON GANGLIOSIDE G-D-3-COATED SURFACES.
AUTHOR(S): MAZUROV A V [Reprint author]; PROKAZOVA N V; MIKHAILENKO I A; MUKHIN D N; REPIN V S; BERGEL'SON L B
CORPORATE SOURCE: ALL-UNION CARDIOL SCI CENT, ACAD MED SCI USSR, MOSCOW, USSR
SOURCE: Doklady Akademii Nauk SSSR, (1987) Vol. 296, No. 5, pp. 1274-1277.
CODEN: DANKAS. ISSN: 0002-3264.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: RUSSIAN
ENTRY DATE: Entered STN: 7 Sep 1988
Last Updated on STN: 7 Sep 1988

AB The interaction between human thrombocytes and ganglioside-covered surfaces was studied with the use of scanning electron microscopy. The following immobilized gangliosides were used: GT1b, GD1a, GM3 and GM1 as well as GD3. Gangliosides GD3, as opposed to other studied gangliosides, contains two **sialic acid** residues at the end of its carbohydrate chain. It is possible that the strong negative charge generated by these residues mediates the stimulating effect of ganglioside GD3 on thrombocytes. Another reason for the stimulating effect of ganglioside GD3 on thrombocytes may be the presence of GD3-specific receptor on the surface of thrombocytes. The fact that ganglioside GD3 stimulates adhesion in thrombocytes and activates them is of great importance due to unusually high levels of this ganglioside in rapidly proliferating smooth muscle intimal cells in **atherosclerosis**.

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(FILE 'HOME' ENTERED AT 08:02:19 ON 28 FEB 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:02:45 ON 28 FEB 2006

L1 517312 S CELL (W) PROLIFERAT?
L2 281202 S ATHEROSCLEROSIS
L3 9346 S L1 AND L2
L4 72799 S SIALIC (W) ACID
L5 31 S L3 AND L4
L6 13 DUP REM L5 (18 DUPLICATES REMOVED)